heart beat was followed by an extrasystole with a compensatory pause. The *a-c* interval was just one-fifth of a second. During the extrasystole there was only one wave in the jugular tracing which was synchronous with the wave in the carotid tracing. This wave was larger than the *c* waves of the normal cardiac cycles and suggested that the auricles and ventricles were in simultaneous contraction. The patient was put on ℥i of tincture of digitalis daily, and this was continued for eight days. After ℥ii of the digitalis the pulse-rate fell to 75 per minute, and apart from an occasional extrasystole the pulse became regular for considerable periods. Chart 1 is an example of this rhythm. Except for an extrasystole occurring at the second cycle the heart's action is perfectly regular. The jugular pulse shows well-marked *a* and *c* waves corresponding to each cardiac cycle. The *a-c* interval is prolonged. The apex-beat tracing shows an unusually well-marked *a* wave preceding the ventricular rise. Chart 2 shows the same rhythm with the occurrence of an extrasystole at the fourth cycle. The *a-c* interval is definitely prolonged. The extrasystole is fully compensatory and the jugular tracing shows only a single wave *c* synchronous with the ventricular rise in the apex-beat tracing. This *c* wave, however, appears at exactly the moment when a normal auricular contraction is due, and from a polygraphic tracing alone it is impossible to say whether the simultaneous contraction of auricles and ventricles is due to a ventricular or an auriculo-ventricular extrasystole. Occasionally two such extrasystoles followed one another, as seen in Chart 3.

In addition to these extrasystoles the heart's action showed the frequent occurrence of the irregularity seen in Chart 4, A and B. In this tracing the first five cycles show, both in the jugular and apex-beat tracings, auricular and ventricular waves with a prolonged *a-c* interval. Following the fifth cycle is an extrasystole of the same type as in Charts 2 and 3. From here onwards we have a coupled rhythm with a gradual diminution in the *a-c* interval of the first beat of the couple till in the fifth and sixth couples the *a-c* interval has completely disappeared, and the auricles and ventricles are contracting simultaneously. At the seventh couple the *a-c* interval again appears. This peculiar rhythm frequently appeared during the patient's stay in hospital, and always presented the same features.

This gradual disappearance of the *a-c* interval can only be attributed to a temporary change in the site of the pace-maker of the heart from the sinus node to some part of the A.-V. node or bundle, probably the result of an irritative lesion in that neighbourhood. As corroborative evidence we have the presence of extrasystoles and a prolonged *a-c* interval. The fact that in the experimental production of an A.-V. rhythm the *a-c* interval shows a gradual shortening and not an abrupt disappearance has been demonstrated by Hering and others. It has been suggested that this gradual change depends either on a gradual shifting or wandering downwards of the pace-maker or to changes in conduction time.

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Aberdeen.

A CASE OF ACHOLURIC JAUNDICE.

BY W. HARMENS, M.B., B.C. CANTAB.

THE following case presents several points of interest both as regards diagnosis and treatment.

The patient is a married woman, aged 27, who has two children, both girls, aged 5½ and 2½ years respectively. In neither is there any enlargement of the spleen. Her maternal grandmother died in rather early life from a "floating tumour" in the abdomen. Her mother died seven years ago from "hæmorrhage," having been in bed a month. Splenic enlargement had been noticed 16 years before her death. During that time she had much ill-health with repeated attacks of jaundice. She was always more or less "yellow." When the patient was 16 years old a swelling was noticed in her left side. Jaundice appeared for the first time 18 months ago, and she lost considerably in weight. She complained of inability to do her work properly, loss of appetite, and feeling generally ill. A considerable amount of pain was present in the left side of a dragging nature, but it was never acute. She was sent to St. Thomas's Hospital about 18 months ago with the diagnosis "splenic enlargement." There she gradually improved under treatment. The spleen extended to a point at the level of the umbilicus. The liver was also slightly enlarged. A moderate degree of anæmia was present. Her blood count at that time was: red cells, 3,000,000; white cells, 9200. An X ray examination was made but was negative.

In the beginning of May, 1914, the same symptoms reappeared. A slight degree of jaundice developed, which gradually became deeper but was never intense. The pain in the left side was nearly always present and often severe, and prevented her carrying on her household duties. She looked pale and anæmic, and said she had lost flesh. There was marked enlargement of the spleen, which extended to a point half an inch below a horizontal line drawn through the umbilicus. No tenderness was present. The liver was slightly enlarged, and extended from the sixth rib above to a point three-quarters of an inch below the costal margin. Hæmic murmurs were present over the heart. The urine was very dark in colour, acid in reaction, of specific gravity 1024, and contained no albumin or sugar. Bile pigment was not present. There were no signs of disease elsewhere. In view of her symptoms I sent her to St. Thomas's Hospital, where she was admitted under Dr. H. G. Turney. On July 9th a differential blood count was made by Dr. L. S. Dudgeon as follows: Erythrocytes, 4,000,000; slight poikilocytosis present; rouleaux formation fair; fibrin formation fair. Hæmoglobin, 90 per cent. Colour index, 1·1. Leucocytes, 10,240; polynuclear neutrophiles, 64·25 per cent.; polynuclear eosinophiles, 2·25 per cent.; small lymphocytes, 25·75 per cent.; large lymphocytes, 3·50 per cent.; large hyaline, 3·25 per cent.; coarsely granular basophiles, 1·00 per cent. No nucleated red cells were seen. The coagulation-time was normal. The fragility of the red cells, tested by sodium chloride in distilled water, was very markedly increased as in congenital cholæmia.

NaCl.	Normal.	Case under discussion.
0·1%	Complete hæmolysis	Complete hæmolysis.
0·2%	,,	,,
0·3%	,,	,,
0·4%	Tinge	,,
0·5%	No hæmolysis	,,
0·6%	,,	Faint tinge.
0·7%	,,	Trace.
0·75%	,,	No hæmolysis.
0·8%	,,	,,

A Wassermann reaction was done but was negative. While in hospital the temperature was slightly raised, 100·2° F. being the highest recorded. The mean was about 99·4°.

For reasons which will presently be discussed it was decided to remove the spleen. The operation was performed on July 27th by Mr. H. Betham Robinson. The anæsthetic used was chloroform and ether. The peritoneum was found to be adherent in many places. The patient stood the operation well, and had the stitches removed on the seventh

day. On August 5th another examination of the blood was made as follows:—Erythrocytes, 3,612,500; rouleaux formation good; fibrin formation good. Hæmoglobin, 75 per cent. Colour index, 1. Leucocytes, 7800; polynuclear neutrophiles, 42·75 per cent.; polynuclear eosinophiles, 2·75 per cent.; small lymphocytes, 32·00 per cent.; large lymphocytes, 9·00 per cent.; large hyaline, 12·00 per cent.; and coarsely granular basophiles, 1·5 per cent. On August 12th, the day of her discharge from hospital, the fragility of the red cells was much less than at the previous examination.

NaCl in distilled water.		
0·1 per cent.	.....	Complete hæmolysis.
0·2 ,,	.....	,,
0·3 ,,	.....	,,
0·35 ,,	.....	,,
0·52 ,,	.....	Faint trace.
0·55 ,,	.....	No hæmolysis.
0·6 ,,	.....	,,

The patient's weight on admission was 7 st. 6 lb. and on her discharge 7 st. 12½ lb. Her colour had greatly improved, and her appetite was good. On Oct. 1st the fragility of the red cells was again tested and was found to be practically normal.

NaCl in distilled water.		
0·38 per cent.	.....	Complete hæmolysis.
0·52 ,,	.....	Trace.
0·6 ,,	.....	No hæmolysis.

For a time after the operation the patient had a slight limp and a dragging pain in the left side. These symptoms have completely disappeared, and she says she feels perfectly well. She is able to carry out her household duties and look after her husband and children. She is delighted with the change in herself since the operation.

Two clinical groups of acholuric jaundice may be recognised: in the one the condition is congenital and hereditary or familial, in the other acquired and lacking the hereditary tendency. In other respects the course and symptoms of the two groups are identical. The case just described seems to be a compromise between the two types. On the one hand the hereditary tendency is very marked, while on the other there was no appearance of symptoms till about the time of puberty. Of course it is possible, as there was never anything like persistent jaundice, that the case was really congenital but latent.

With regard to the indication for operation, it must be granted that the mere diagnosis of acholuric jaundice is not in itself sufficient. The condition, whether congenital or acquired, is compatible with many years of healthy life. In this patient the impairment of general health was very considerable. Abdominal pains were more or less persistent. They were due probably to the dragging of the splenic tumour so far as the more chronic ones were concerned, but in addition to these there were others of a different type and of a different distribution which occurred synchronously with the attacks of jaundice, and were dependent to all seeming upon changes in the gall-bladder and ducts. These symptoms in themselves afforded sufficient justification for the removal of the spleen, especially as the organ was of moderate size and its extirpation promised to be attended with the minimum of risk. But in addition to these actual inconveniences from which the patient might be promised relief there were others looming in the future which could not be ignored. The mother died from hæmatemesis 16 years after the enlargement of the spleen was diagnosed, and there is no ground for suspecting in her case either alcoholic excess or the presence of a gastric ulcer. It is quite certain that the exaggerated hæmolysis which is the underlying condition of acholuric jaundice must throw an enormous increase of work upon the liver, and there is clinical evidence of this in the enlargement of that organ. Eppinger has shown that even during apparently quiescent periods of acholuric jaundice the excretion of urobilinogen may be increased ten- or even twenty-fold, and increase of function to this extent must surely entail some danger of structural change in the long run in the organs concerned. The history of the mother's illness is, to say the least, suggestive of the same sequence of pathological events that we find in Banti's disease, and in both mother and daughter we find the same history of repeated attacks of jaundice with enlargement of spleen. So far as I am aware, the only change hitherto described as occurring in the liver in these cases is the accumulation of pigment-bearing cells without cirrhosis, but pathological evidence of any sort is far too scanty for a final verdict.

I wish to acknowledge the great kindness of Dr. Turney in the help he has given me, and to thank both him and Dr. Dudgeon for the use of the notes on the case.

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CEREBRO-SPINAL MENINGITIS: MENINGOCOCCI FOUND IN PERIPHERAL BLOOD FILMS.

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Dr. A. D. Edwards, medical officer of health of Bournemouth, has given me the following notes of this septicæmic type of case of cerebro-spinal meningitis.

The patient, a male aged 33, a carpenter, was at work on the morning of March 15th, when he complained of pains in the lower part of the back, felt shivery, and soon afterwards vomited. After this sudden onset the patient became rapidly worse, and by the evening he complained of "aching all over." A purpuric rash appeared on the following morning on the brow and face. Retraction of the head was not present at this or any other stage of the illness. Dr. Edwards saw the patient for the first time on the afternoon of March 16th when his temperature was normal, but the pulse could not be felt. There was a well-marked purpuric rash on the face consisting mainly of more or less circular spots, dark purple in colour. There was also extravasation of blood on the trunk, especially on the buttocks. The patient was conscious and complained of "pains all over," so that he could not rest easily in bed. Pains in the knees and elbows were marked, especially on movement; for this reason Kernig's sign was not available. On lumbar puncture 25 c.c. of cerebro-spinal fluid were removed, and antimeningococcic serum was injected to the same quantity. The fluid came out slowly; it was mixed with blood, but otherwise was not very turbid. No growth occurred on blood serum or on agar, although there was blood in the inoculating fluid. The patient's temperature rose on the night of March 16th to 102° F., but fell on the morning of the 17th. There was diarrhœa on the night of March 16th, and the vomiting which had been occasional became frequent on the morning of the 17th. During that morning the patient became comatose and died on the afternoon of the 17th, 50 hours after the onset of the first symptom.

Clinically the case appeared to be a picture of profound septicæmia; there were no signs or symptoms to indicate the presence of meningitis. This latter condition was suspected mainly by the process of exclusion, and because the epidemic disease was known to be prevalent in certain military centres at the present time.

On March 17th, at 11.30 A.M., I made films from the blood obtained by puncturing the ear and